

=> Biotinylation
L1 3322 BIOTINYLATION

=> "viral antigen"
L2 9232 "VIRAL ANTIGEN"

=> L1 and L2
L3 3 L1 AND L2

=> "viral envelope protein"
L4 1012 "VIRAL ENVELOPE PROTEIN"

=> L1 and L4
L5 1 L1 AND L4

=> "viral surface protein"
L6 138 "VIRAL SURFACE PROTEIN"

=> L1 and L6
L7 0 L1 AND L6

=> influenza and L1
L8 19 INFLUENZA AND L1

=> HIV and L1
L9 54 HIV AND L1

=> gp120 and L9
L10 7 GP120 AND L9

=> gp160 and L1
L11 10 GP160 AND L1

=> D L3 IBIB TI AU ABS 1-

DOCUMENT NUMBER: PREV199497549929
TITLE: Complete inactivation of target mRNA by biotinylated antisense oligodeoxynucleotide-avidin conjugates.
AUTHOR(S): Boado, Ruben J. [Reprint author]; Pardridge, William M.
CORPORATE SOURCE: Dep. Med., Brain Res. Inst., UCLA Sch. Med., Los Angeles, CA 90024, USA
SOURCE: Bioconjugate Chemistry, (1994) Vol. 5, No. 5, pp. 406-410. CODEN: BCCHES. ISSN: 1043-1802.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 15 Dec 1994
Last Updated on STN: 15 Dec 1994

TI Complete inactivation of target mRNA by biotinylated antisense oligodeoxynucleotide-avidin conjugates.
SO Bioconjugate Chemistry, (1994) Vol. 5, No. 5, pp. 406-410. CODEN: BCCHES. ISSN: 1043-1802.
AU Boado, Ruben J. [Reprint author]; Pardridge, William M.
AB **Biotinylation** of phosphodiester oligodeoxynucleotides (PO-ODN) allows for conjugation to avidin-based transcellular delivery systems. In addition, **biotinylation** of PO-ODN at the 3'-terminus provides complete protection against serum 3'-exonuclease degradation. The present study was undertaken to determine if antisense 3'-biotinylated PO-ODN-avidin constructs are able to recognize and inactivate the target mRNA through RNase H-mediated degradation. A 21-mer antisense PO-ODN complementary to the tat gene encompassing nucleotides 5402-5422 of the **HIV-1** genome was synthesized with biotin conjugated to the 3'-terminus (bio-tat). Gel mobility assays using (5'-32P)-labeled bio-tat ODN and avidin showed that the bio-tat ODN was fully monobiotinylated. Aliquots of (32P)-labeled sense or antisense tat RNA (337 and 351 nucleotides, respectively) were prepared from transcription plasmids and were preincubated with an excess of bio-tat ODN with or without avidin constructs and digested with RNase H. Products were resolved with sequencing gel and analyzed by autoradiography. Complete conversion to predicted RNA fragments resulting from RNase H digestion of the RNA-ODN duplex (53 and 263 nucleotides) was observed when (32P)-tat sense RNA was incubated with antisense bio-tat ODN or conjugated to avidin or an avidin-cationized human serum albumin (cHSA) complex. Conversely, no degradation of (32P)-tat-antisense RNA was observed after incubation with antisense bio-tat ODN and RNase H. In addition, the avidin-cHSA complex significantly increased (84-fold) the uptake of (32P)-internally labeled bio-tat ODN and its stability against cellular nuclease degradation in peripheral blood lymphocytes. In conclusion, biotinylated antisense ODN-avidin constructs induce complete inactivation of target mRNA by RNase H. Therefore, 3'-biotinylated PO-ODNs have the advantages of (a) resistance to serum and cellular 3'-exonuclease, (b) conjugation by avidin-based transcellular delivery systems, and (c) inactivation of target mRNA via RNase H degradation.

L9 ANSWER 48 OF 54 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 1998:406124 BIOSIS
DOCUMENT NUMBER: PREV199800406124
TITLE: Retention of the human immunodeficiency virus type 1
envelope glycoprotein in the endoplasmic reticulum does not
redirect virus assembly from the plasma membrane.
AUTHOR(S): Salzwedel, Karl; West, John T., Jr.; Mulligan, Mark J.;
Hunter, Eric [Reprint author]
CORPORATE SOURCE: Dep. Microbiol., Univ. Alabama at Birmingham, 845 19th St.
South, Birmingham, AL 35294-2170, USA
SOURCE: Journal of Virology, (Sept., 1998) Vol. 72, No. 9, pp.
7523-7531. print.
CODEN: JOVIAM. ISSN: 0022-538X.

DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 21 Sep 1998
Last Updated on STN: 21 Sep 1998

TI Retention of the human immunodeficiency virus type 1 envelope glycoprotein
in the endoplasmic reticulum does not redirect virus assembly from the
plasma membrane.
SO Journal of Virology, (Sept., 1998) Vol. 72, No. 9, pp. 7523-7531. print.
CODEN: JOVIAM. ISSN: 0022-538X.
AU Salzwedel, Karl; West, John T., Jr.; Mulligan, Mark J.; Hunter, Eric
[Reprint author]
AB The envelope glycoprotein (Env) of human immunodeficiency virus type 1 (**HIV-1**) has been shown to redirect the site of virus assembly in
polarized epithelial cells. To test whether localization of the
glycoprotein exclusively to the endoplasmic reticulum (ER) could redirect
virus assembly to that organelle in nonpolarized cells, an ER -retrieval
signal was engineered into an epitope-tagged variant of Env. The epitope
tag, attached to the C terminus of Env, did not affect the normal
maturation and transport of the glycoprotein or the incorporation of Env
into virions. The epitope-tagged Env was also capable of mediating
syncytium formation and virus entry with a similar efficiency to that of
wild-type Env. When the epitope was modified to contain a consensus
K(X)KXX ER retrieval signal, however, the glycoprotein was no longer
proteolytically processed into its surface and transmembrane subunits and
Env could not be detected at the cell surface by **biotinylation**.
Endoglycosidase H analysis revealed that the modified Env was not
transported to the Golgi apparatus. Immunofluorescent staining patterns
were also consistent with the exclusion of Env from the Golgi. As
expected, cells expressing the modified Env failed to form syncytia with
CD4+ permissive cells. Despite this tight localization of Env to the ER,
when the modified Env was expressed in the context of virus, virions
continued to be produced efficiently from the plasma membrane of
transfected cells. However, these virions contained no detectable
glycoprotein and were noninfectious. Electron microscopy revealed virus
budding from the plasma membrane of these cells, but no virus was seen
assembling at the ER membrane and no assembled virions were found within
the cell. These results suggest that the accumulation of Env in an
intracellular compartment is not sufficient to redirect the assembly of
HIV Gag in nonpolarized cells.

L9 ANSWER 30 OF 54 CAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 1994:506400 CAPLUS

DOCUMENT NUMBER: 121:106400

TITLE: Human immunodeficiency virus type 1 Nef-induced down-modulation of CD4 is due to rapid internalization and degradation of surface CD4

AUTHOR(S): Rhee, Sung S.; Marsh, Jon W.

CORPORATE SOURCE: Laboratory of Molecular Biology, Natl. Institute of Mental Health, Bethesda, MD, 20892, USA

SOURCE: Journal of Virology (1994), 68(8), 5156-63

CODEN: JOVIAM; ISSN: 0022-538X

DOCUMENT TYPE: Journal

LANGUAGE: English

TI Human immunodeficiency virus type 1 Nef-induced down-modulation of CD4 is due to rapid internalization and degradation of surface CD4

SO Journal of Virology (1994), 68(8), 5156-63

CODEN: JOVIAM; ISSN: 0022-538X

AU Rhee, Sung S.; Marsh, Jon W.

AB Human immunodeficiency virus type 1 (**HIV-1**) Nef is a myristoylated protein with a relative mol. mass of 27 kDa, is localized to the cytoplasmic surfaces of cellular membranes, and has been reported to down-modulate CD4 in human T cells. To understand the mechanism of **HIV-1** Nef-mediated down-modulation of cell surface CD4, the authors expressed Nef protein in human T-cell line VB. Expression of **HIV-1** Nef protein down-modulated surface CD4 mols. In pulse-chase expts., CD4 mols. in Nef-expressing cells were synthesized at normal levels. However, the bulk of newly synthesized CD4 protein was degraded with a half-life of approx. 6 h, compared with the 24-h half-life in control cells. This Nef-induced acceleration of CD4 turnover was inhibited by lysosomotropic agents NH₄Cl and chloroquine as well as by the protease inhibitor leupeptin. Surface CD4 **biotinylation** expts. demonstrated that CD4 mols. in Nef-expressing T cells are transported to the plasma membrane with normal kinetics but are then rapidly internalized. Therefore, **HIV-1** Nef-induced down-modulation of CD4 is due to rapid internalization of surface CD4 and subsequent degradation by an acid-dependent process, potentially lysosomal. Addnl., in a Nef-expressing cell, the authors find accelerated dissociation of the T-cell tyrosine kinase p56lck and CD4 but only after the complex reaches the plasma membrane. This implies that **HIV-1** Nef protein might play a role in triggering a series of T-cell activation-like events, which contribute to p56lck dissociation and internalization of surface CD4 mols.

L9 ANSWER 28 OF 54 CAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 1994:628630 CAPLUS

DOCUMENT NUMBER: 121:228630

TITLE: Altered glycosylation of leukosialin, CD43, in
HIV-1-infected cells of the CEM line

AUTHOR(S): Lefebvre, Jean-Claude; Giordanengo, Valerie; Limouse,
Martine; Doglio, Alain; Cucchiaroni, Magali; Monpoux,
Fabrice; Mariani, Roger; Peyron, Jean-Francois
CORPORATE SOURCE: Lab. Virol., Unite Inst. Natl. Sante Rech. Med.
(INSERM), Nice, 06107, Fr.

SOURCE: Journal of Experimental Medicine (1994), 180(5),
1609-17

CODEN: JEMEAV; ISSN: 0022-1007

DOCUMENT TYPE: Journal

LANGUAGE: English

TI Altered glycosylation of leukosialin, CD43, in **HIV-1**-infected
cells of the CEM line

SO Journal of Experimental Medicine (1994), 180(5), 1609-17
CODEN: JEMEAV; ISSN: 0022-1007

AU Lefebvre, Jean-Claude; Giordanengo, Valerie; Limouse, Martine; Doglio,
Alain; Cucchiaroni, Magali; Monpoux, Fabrice; Mariani, Roger; Peyron,
Jean-Francois

AB CD43 (leukosialin, gpL115, sialophorin) is a major sialoglycoprotein
widely expressed on hematopoietic cells that is defective in the
congenital immunodeficiency Wiskott-Aldrich syndrome. It is thought to
play an important role in cell-cell interactions and to be a costimulatory
mol. for T lymphocyte activation. Using a metabolic ³⁵S042- radiolabeling
assay or **biotinylation** of cell surface proteins, the authors
discovered that CD43 are sulfated mols. the glycosylation of which is
altered in human immunodeficiency virus type 1 (**HIV-1**)-infected
leukemic T cells of the CEM line. Hyposialylation of O-glycans and
changed substitution on N-acetylgalactosamine residues are observed The
glycosylation defect is associated with an impairment of CD43-mediated
homotypic aggregation which can be restored by resialylation. The
hyposialylation of CD43 on **HIV-1**+ cells may explain the high
prevalence of autoantibodies directed against nonsialylated CD43 that have
been detected in **HIV-1**-infected individuals. A defect in
glycosylation of important mols. such as CD43 or, as recently described,
CD45 may explain alterations of T cell functions and viability in
HIV-1-infected individuals. In addition, a possible implication of
hyposialylation in the **HIV-1**-infected cells entrapment in lymph
nodes could be envisioned.

ACCESSION NUMBER: 1998:540537 CAPLUS
DOCUMENT NUMBER: 129:257507
TITLE: Retention of the human immunodeficiency virus type 1 envelope glycoprotein in the endoplasmic reticulum does not redirect virus assembly from the plasma membrane
AUTHOR(S): Salzwedel, Karl; West, John T., Jr.; Mulligan, Mark J.; Hunter, Eric
CORPORATE SOURCE: Department of Microbiology, University of Alabama at Birmingham, Birmingham, AL, 35294, USA
SOURCE: Journal of Virology (1998), 72(9), 7523-7531
CODEN: JOVIAM; ISSN: 0022-538X
PUBLISHER: American Society for Microbiology
DOCUMENT TYPE: Journal
LANGUAGE: English

TI Retention of the human immunodeficiency virus type 1 envelope glycoprotein in the endoplasmic reticulum does not redirect virus assembly from the plasma membrane
SO Journal of Virology (1998), 72(9), 7523-7531
CODEN: JOVIAM; ISSN: 0022-538X
AU Salzwedel, Karl; West, John T., Jr.; Mulligan, Mark J.; Hunter, Eric
AB The envelope glycoprotein (Env) of human immunodeficiency virus type 1 (HIV-1) has been shown to redirect the site of virus assembly in polarized epithelial cells. To test whether localization of the glycoprotein exclusively to the endoplasmic reticulum (ER) could redirect virus assembly to that organelle in nonpolarized cells, an ER-retrieval signal was engineered into an epitope-tagged variant of Env. The epitope tag, attached to the C terminus of Env, did not affect the normal maturation and transport of the glycoprotein or the incorporation of Env into virions. The epitope-tagged Env was also capable of mediating syncytium formation and virus entry with a similar efficiency to that of wild-type Env. When the epitope was modified to contain a consensus K(X)KXX ER retrieval signal, however, the glycoprotein was no longer proteolytically processed into its surface and transmembrane subunits and Env could not be detected at the cell surface by **biotinylation**. Endoglycosidase H anal. revealed that the modified Env was not transported to the Golgi apparatus. Immunofluorescent staining patterns were also consistent with the exclusion of Env from the Golgi. As expected, cells expressing the modified Env failed to form syncytia with CD4+ permissive cells. Despite this tight localization of Env to the ER, when the modified Env was expressed in the context of virus, virions continued to be produced efficiently from the plasma membrane of transfected cells. However, these virions contained no detectable glycoprotein and were noninfectious. Electron microscopy revealed virus budding from the plasma membrane of these cells, but no virus was seen assembling at the ER membrane and no assembled virions were found within the cell. These results suggest that the accumulation of Env in an intracellular

ACCESSION NUMBER: 1999:167527 CAPLUS
 DOCUMENT NUMBER: 131:70988
 TITLE: Processing and routage of **HIV** glycoproteins
 by furin to the cell surface
 AUTHOR(S): Moulard, Maxime; Hallenberger, Sabine; Garten,
 Wolfgang; Klenk, Hans-Dieter
 CORPORATE SOURCE: Centre d'Immunologie de Marseille Luminy, Marseille,
 Fr.
 SOURCE: Virus Research (1999), 60(1), 55-65
 CODEN: VIREDF; ISSN: 0168-1702
 PUBLISHER: Elsevier Science Ireland Ltd.
 DOCUMENT TYPE: Journal
 LANGUAGE: English

TI Processing and routage of **HIV** glycoproteins by furin to the cell
 surface
 SO Virus Research (1999), 60(1), 55-65
 CODEN: VIREDF; ISSN: 0168-1702
 AU Moulard, Maxime; Hallenberger, Sabine; Garten, Wolfgang; Klenk, Hans-Dieter
 AB Proteolytic activation of **HIV**-1 and **HIV**-2 envelope
 glycoprotein precursors (gp160 and gp140, resp.) occurs at the carboxyl
 side of a consensus motif consisting of the highly basic amino acid
 sequence. We have shown previously () and confirmed in this report, that
 furin and PC7 can be considered as the putative physiol. enzymes involved
 in the proteolytic activation of the **HIV**-1 and **HIV**-2
 envelope precursors. In this study, we show by cell surface
biotinylation and immunopptn. of the cell surface associated viral
 glycoproteins with antibodies that the mature viral envelope glycoproteins
 are correctly transported to the cell membrane. Furthermore, we show that
 the uncleaved forms of the glycoproteins (gp160HIV-1 and gp140HIV-2) are
 also highly represented at the cell surface. First, transient expression
 of gp160 and gp140 into CV1, a cell line known to be inefficient in the
 proteolytic processing of the env gene, results in the expression of gp160
 and gp140 at the cell surface. Moreover, **HIV**-1 infection of T
 cells also showed that gp160 is directed to the cell surface. In addition,
 the authors show that the precursor is not incorporated in the virus
 particle following the budding from the cell surface. Furthermore, a
 gp160 mutant (deficient for three carbohydrate sites on the gp41), shown
 to be poorly processed with the coexpressed endoproteases, is found to be
 transported as an uncleaved precursor to the cell surface. In contrast to
HIV envelope glycoproteins, the influenza hemagglutinin precursor
 (HA0), that is thought to be matured by the furin-like enzymes as well, is
 found to be retained within the cell and is not able to reach the cell
 surface. These results show that the proteolytic maturation of the viral
 envelope precursors of human immunodeficiency viruses type 1 and type 2 is
 not a prerequisite for cell surface targeting of the **HIV**
 glycoproteins. Implications of these results for antiviral immune
 response are discussed.

L8 ANSWER 15 OF 19 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
 ACCESSION NUMBER: 1999:47203 BIOSIS
 DOCUMENT NUMBER: PREV199900047203
 TITLE: Use of the ARPE-19 cell line as a model of RPE polarity:
 Basolateral secretion of FGF5.
 AUTHOR(S): Dunn, Kerrin C.; Marmorstein, Alan D.; Bonilba, Vera L.;
 Rodriguez-Boulan, Enrique; Giordano, Frank; Hjelmeland,
 Leonard M. [Reprint author]
 CORPORATE SOURCE: Vitreoretinal Res. Lab., Sch. Med., Univ. Calif., One
 Shields Ave., Davis, CA 95616-8794, USA
 SOURCE: IOVS, (Dec., 1998) Vol. 39, No. 13, pp. 2744-2749. print.
 DOCUMENT TYPE: Article
 LANGUAGE: English
 ENTRY DATE: Entered STN: 10 Feb 1999
 Last Updated on STN: 10 Feb 1999

TI Use of the ARPE-19 cell line as a model of RPE polarity: Basolateral
 secretion of FGF5.
 SO IOVS, (Dec., 1998) Vol. 39, No. 13, pp. 2744-2749. print.
 AU Dunn, Kerrin C.; Marmorstein, Alan D.; Bonilba, Vera L.; Rodriguez-Boulan,
 Enrique; Giordano, Frank; Hjelmeland, Leonard M. [Reprint author]
 AB PURPOSE. To determine the polarity of fibroblast growth factor 5 (FGF5)
 secretions from retinal pigment epithelium (RPE) cells and to examine the
 viability and utility of the ARPE-19 cell line as a model for the study of
 RPE polarity. METHODS. **Influenza** infection and
 adenovirus-mediated gene transfer were used to deliver and express genes
 encoding **influenza** hemagglutinin (HA), p75-NTR (a neurotrophin
 receptor), low-density lipoprotein (LDL) receptor (LDLR), and FGF5 in
 confluent monolayers of ARPE-19 cells. The localization of HA, p75-NTR,
 and LDLR was determined by confocal microscopy. Domain selective
biotinylation assays were used to quantitatively determine the
 polarities of p75-NTR and LDLR. The secretion of FGF5 into the apical and
 basal media of ARPE-19 cultures was examined by immunoblot analysis of
 conditioned media. RESULTS. Hemagglutinin and p75-NTR were found to be
 localized on the apical surface of infected and transduced ARPE-19 cells.
 In contrast, LDLR was associated preferentially with the basolateral
 membrane of ARPE-19 cells. **Biotinylation** studies indicated that
 84% of p75-NTR was present on the apical surface, and 79% of LDLR was
 basolaterally polarized. Over the course of 6 hours, more than 90% of the
 total secreted FGF5 protein accumulated in the basolateral media.
 CONCLUSIONS. ARPE-19 cells exhibit a polarized distribution of cell
 surface markers when examined by either confocal microscopy or
 surface-labeling assays. This indicates that the ARPE-19 cell line is a
 valid model for studies of RPE cell polarity. FGF5, a secreted protein
 normally produced by RPE cells, is accumulated preferentially in the basal
 media after only 6 hours, suggesting that it is vectorially secreted from
 the basolateral surface of ARPE-19 cells.

L8 ANSWER 13 OF 19 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 1999:391802 BIOSIS
DOCUMENT NUMBER: PREV199900391802
TITLE: Polarity of osteoblasts and osteoblast-like UMR-108 cells.
AUTHOR(S): Ilvesaro, Joanna [Reprint author]; Metsikko, Kalervo;
Vaananen, Kalervo; Tuukkanen, Juha
CORPORATE SOURCE: Department of Anatomy, University of Oulu, Kajaanintie 52A,
FIN-90220, Oulu, Finland
SOURCE: Journal of Bone and Mineral Research, (Aug., 1999) Vol. 14,
No. 8, pp. 1338-1344. print.
CODEN: JBMREJ. ISSN: 0884-0431.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 28 Sep 1999
Last Updated on STN: 28 Sep 1999

TI Polarity of osteoblasts and osteoblast-like UMR-108 cells.
SO Journal of Bone and Mineral Research, (Aug., 1999) Vol. 14, No. 8, pp.
1338-1344. print.
CODEN: JBMREJ. ISSN: 0884-0431.
AU Ilvesaro, Joanna [Reprint author]; Metsikko, Kalervo; Vaananen, Kalervo;
Tuukkanen, Juha
AB Enveloped viruses, such as vesicular stomatitis virus (VSV) and
Influenza virus, have been widely used in studying epithelial cell
polarity. Viral particles of VSV-infected epithelial cells bud from the
basolateral membrane, which is in contact with the internal milieu and the
blood supply. **Influenza**-infected cells bud viral particles from
the apical surface facing the external milieu. This feature can be
utilized in labeling polarized membrane domains. We studied the polarity
of mesenchymal osteoblasts using osteosarcoma cell line UMR-108 and
endosteal osteoblasts in situ in bone tissue cultures. Immunofluorescence
confocal microscopy revealed that the VSV glycoprotein (VSV G) was
targeted to the culture medium-facing surface. In endosteal osteoblasts,
VSV G protein was found in the surface facing bone marrow and circulation.
On the contrary, **Influenza** virus hemagglutinin (HA) was
localized to the bone substrate-facing surface of the UMR-108 cells.
Electron microscopy showed that in the cases where the cells were growing
as a single layer, VSV particles were budding from the culture
medium-facing surface, whereas **Influenza** viruses budded from the
bone substrate-facing surface. When the cells overlapped, this polarity
was lost. Cell surface **biotinylation** revealed that 55% of VSV G
protein was biotinylated, whereas **Influenza** virus HA was only
22% biotinylated. These findings suggest that osteoblasts are polarized
at some point of their life cycle. The bone-attaching plasma membrane of
osteoblasts is apical, and the circulation or bone marrow-facing plasma
membrane is basolateral in nature.

L8 ANSWER 12 OF 19 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 2000:122577 BIOSIS
DOCUMENT NUMBER: PREV200000122577
TITLE: MAL, an integral element of the apical sorting machinery,
is an itinerant protein that cycles between the trans-Golgi
network and the plasma membrane.
AUTHOR(S): Puertollano, Rosa; Alonso, Miguel A. [Reprint author]
CORPORATE SOURCE: Centro de Biologia Molecular "Severo Ochoa," Consejo
Superior de Investigaciones Cientificas, Universidad
Autonoma de Madrid, Cantoblanco, 28049, Madrid, Spain
SOURCE: Molecular Biology of the Cell, (Oct., 1999) Vol. 10, No.
10, pp. 3435-3447. print.
CODEN: MBCEEV. ISSN: 1059-1524.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 5 Apr 2000
Last Updated on STN: 3 Jan 2002

TI MAL, an integral element of the apical sorting machinery, is an itinerant
protein that cycles between the trans-Golgi network and the plasma
membrane.

SO Molecular Biology of the Cell, (Oct., 1999) Vol. 10, No. 10, pp.
3435-3447. print.
CODEN: MBCEEV. ISSN: 1059-1524.

AU Puertollano, Rosa; Alonso, Miguel A. [Reprint author]

AB The MAL proteolipid is a nonglycosylated integral membrane protein found
in glycolipid-enriched membrane microdomains. In polarized epithelial
Madin-Darby canine kidney cells, MAL is necessary for normal apical
transport and accurate sorting of the **influenza** virus
hemagglutinin. MAL is thus part of the integral machinery for
glycolipid-enriched membrane-mediated apical transport. At steady state,
MAL is predominantly located in perinuclear vesicles that probably arise
from the trans-Golgi network (TGN). To act on membrane traffic and to
prevent their accumulation in the target compartment, integral membrane
elements of the protein-sorting machinery should be itinerant proteins
that cycle between the donor and target compartments. To establish
whether MAL is an itinerant protein, we engineered the last extracellular
loop of MAL by insertion of sequences containing the FLAG epitope or with
sequences containing residues that became O-glycosylated within the cells
or that displayed biotinylatable groups. The ectopic expression of these
modified MAL proteins allowed us to investigate the surface expression of
MAL and its movement through different compartments after internalization
with the use of a combination of assays, including surface
biotinylation, surface binding of anti-FLAG antibodies,
neuraminidase sensitivity, and drug treatments. Immunofluorescence and
flow cytometric analyses indicated that, in addition to its Golgi
localization, MAL was also expressed on the cell surface, from which it
was rapidly internalized. This retrieval implies transport through the
endosomal pathway and requires endosomal acidification, because it can be
inhibited by drugs such as chloroquine, monensin, and NH₄Cl.
Resialylation experiments of surface MAL treated with neuraminidase
indicated that apprx30% of the internalized MAL molecules were delivered
to the TGN, probably to start a new cycle of cargo transport. Together,
these observations suggest that, as predicted for integral membrane
members of the late protein transport machinery, MAL is an itinerant
protein cycling between the TGN and the plasma membrane.

L8 ANSWER 10 OF 19 CAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 1992:211876 CAPLUS

DOCUMENT NUMBER: 116:211876

TITLE: Opposite polarity of virus budding and of viral envelope glycoprotein distribution in epithelial cells derived from different tissues

AUTHOR(S): Zurzolo, Chiara; Polistina, Claudio; Saini, Marco; Gentile, Raffaele; Aloj, Luigi; Migliaccio, Giovanni; Bonatti, Stefano; Nitsch, Lucio

CORPORATE SOURCE: Cent. Endocrinol. Oncol. Sper., Cons. Naz. Ric., Naples, 80131, Italy

SOURCE: Journal of Cell Biology (1992), 117(3), 551-64
CODEN: JCLBA3; ISSN: 0021-9525

DOCUMENT TYPE: Journal

LANGUAGE: English

TI Opposite polarity of virus budding and of viral envelope glycoprotein distribution in epithelial cells derived from different tissues

SO Journal of Cell Biology (1992), 117(3), 551-64
CODEN: JCLBA3; ISSN: 0021-9525

AU Zurzolo, Chiara; Polistina, Claudio; Saini, Marco; Gentile, Raffaele; Aloj, Luigi; Migliaccio, Giovanni; Bonatti, Stefano; Nitsch, Lucio

AB The surface envelope glycoprotein distribution and the budding polarity of four RNA viruses in Fischer rat thyroid (FRT) cells and in CaCo-2 cells derived from a human colon carcinoma were compared. Whereas both FRT and CaCo-2 cells sort similarly **influenza** hemagglutinin and vesicular stomatitis virus (VSV) G protein, resp., to apical and basolateral membrane domains, they differ in their handling of two togaviruses Sindbis and Semiliki Forest virus (SFV). By conventional FM, Sindbis virus and SFV bud apically in FRT cells and basolaterally in CaCo-2 cells. Consistent with this finding, the distribution of the p62/E2 envelope glycoprotein of SFV, assayed by immuno-electron microscopy and by domain-selective surface **biotinylation** was predominantly apical on FRT cells and basolateral on CaCo-2 cells. A given virus and its envelope glycoprotein can be delivered to opposite membrane domains in epithelial cells derived from different tissues. The tissue specificity in the polarity of virus budding and viral envelope glycoprotein distribution indicate that the sorting machinery varies considerably between different epithelial cell types.

L10 ANSWER 6 OF 7 CAPLUS COPYRIGHT 2004 ACS on STN

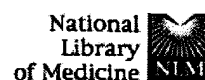
ACCESSION NUMBER: 1994:161617 CAPLUS
DOCUMENT NUMBER: 120:161617
TITLE: Process for the determination of peptides
corresponding to immunologically important epitopes
and their use in a process for determination of
antibodies, or biotinylated peptides corresponding to
immunologically important epitopes, a process for
preparing them and compositions containing them
INVENTOR(S): De Leys, Robert
PATENT ASSIGNEE(S): N.V. Innogenetics S.A., Belg.
SOURCE: PCT Int. Appl., 133 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9318054	A2	19930916	WO 1993-EP517	19930308
WO 9318054	A3	19940217		
W:	AU, BB, BG, BR, CA, CZ, FI, HU, JP, KP, KR, LK, MG, MN, MW, NO, NZ, PL, PT, RO, RU, SD, SK, UA, US			
RW:	AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, SN, TD, TG			
EP 564746	A1	19931013	EP 1992-400598	19920306
R:	BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE			
CA 2102301	AA	19930907	CA 1993-2102301	19930308
AU 9337463	A1	19931005	AU 1993-37463	19930308
AU 671623	B2	19960905		
EP 589004	A1	19940330	EP 1993-906490	19930308
EP 589004	B1	19990506		
R:	AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE			
JP 06505806	T2	19940630	JP 1993-515334	19930308
JP 3443809	B2	20030908		
BR 9305435	A	19941227	BR 1993-5435	19930308
EP 891982	A2	19990120	EP 1998-202777	19930308
EP 891982	A3	20000412		
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AT 179716	E	19990515	AT 1993-906490	19930308
ES 2133392	T3	19990916	ES 1993-906490	19930308
US 5891640	A	19990406	US 1993-146028	19931122
US 6165730	A	20001226	US 1996-723425	19960930
US 6210903	B1	20010403	US 1998-112206	19980709
US 6667387	B1	20031223	US 2000-576824	20000523
US 6649735	B1	20031118	US 2001-790497	20010223
JP 2004002379	A2	20040108	JP 2003-107716	20030411
PRIORITY APPLN. INFO.:			EP 1992-400598	A 19920306
			EP 1993-906490	A3 19930308
			JP 1993-515334	A3 19930308
			WO 1993-EP517	A 19930308
			US 1993-146028	A3 19931122
			US 1996-723425	A3 19960930
			US 2000-576824	A3 20000523
TI	Process for the determination of peptides corresponding to immunologically important epitopes and their use in a process for determination of antibodies, or biotinylated peptides corresponding to immunologically important epitopes, a process for preparing them and compositions containing them			
SO	PCT Int. Appl., 133 pp. CODEN: PIXXD2			
IN	De Leys, Robert			

AB Peptides corresponding to immunol. important epitopes (of bacterial or viral proteins) are determined by (1) preparing peptides corresponding to fragments of the protein of interest, (2) biotinylating the peptides, (3) binding the biotinylated peptides to a solid phase via interaction with avidin or streptavidin, and (4) measuring antibodies which bind to the individual peptides. Processes for **biotinylation** of the peptides and for determination of antibodies to hepatitis C virus (HCV), to **HIV**, and to HTLV-I and -II are also disclosed. HCV, **HIV**, HTLV-I, and HTLV-II peptide sequences are included. Use of the biotinylated peptides in the process of the invention makes the anchorage of the peptides to a solid support such that it leaves their essential amino acids free to be recognized by antibodies. In studies determining binding of unbiotinylated peptides directly onto the wells of a polystyrene microtiter plate and binding of biotinylated peptides to wells coated with streptavidin, results demonstrated that antibody binding to the biotinylated peptide is superior to antibody binding to peptide coated directly onto the plastic

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DOCUMENT NUMBER: PREV200100186731
TITLE: Mutations in the cytoplasmic tail of murine leukemia virus
envelope protein suppress fusion inhibition by R peptide.
AUTHOR(S): Li, Min; Yang, Chinglai; Compans, Richard W. [Reprint
author]
CORPORATE SOURCE: Department of Microbiology and Immunology, Emory University
School of Medicine, Atlanta, GA, 30322, USA
compans@microbio.emory.edu
SOURCE: Journal of Virology, (March, 2001) Vol. 75, No. 5, pp.
2337-2344. print.
CODEN: JOVIAM. ISSN: 0022-538X.
DOCUMENT TYPE: Article
LANGUAGE: English
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TI Mutations in the cytoplasmic tail of murine leukemia virus envelope
protein suppress fusion inhibition by R peptide.
SO Journal of Virology, (March, 2001) Vol. 75, No. 5, pp. 2337-2344. print.
CODEN: JOVIAM. ISSN: 0022-538X.
AU Li, Min; Yang, Chinglai; Compans, Richard W. [Reprint author]
AB During viral maturation, the cytoplasmic tail of the murine leukemia virus
(MuLV) envelope (Env) protein undergoes proteolytic cleavage by the viral
protease to release the 16-amino-acid R peptide, and this cleavage event
activates the Env protein's fusion activity. We introduced Gly and/or Ser
residues at different positions upstream of the R peptide in the
cytoplasmic tail of the Friend MuLV Env protein and investigated their
effects on fusion activity. Expression in HeLa T4 cells of a mutant Env
protein with a single Gly insertion after I619, five amino acids upstream
from the R peptide, induced syncytium formation with overlaid XC cells.
Env proteins containing single or double Gly-Ser insertions after F614, 10
amino acids upstream from the R peptide, induced syncytium formation, and
mutant proteins with multiple Gly insertions induced various levels of
syncytium formation between HeLa T4 and XC cells. Immunoprecipitation and
surface **biotinylation** assays showed that most of the mutants had
surface expression levels comparable to those of the wild-type or R
peptide-truncated Env proteins. Fluorescence dye redistribution assays
also showed no hemifusion in the Env proteins which did not induce fusion.
Our results indicate that insertion mutations in the cytoplasmic tail of
the MuLV Env protein can suppress the inhibitory effect of the R peptide
on membrane fusion and that there are differences in the effects of
insertions in two regions in the cytoplasmic tail upstream of the R
peptide.



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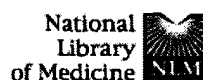
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